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AN ACUTE-TRAUMA INDEX

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May 1977

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Thirty-nine parameters commonly measured when a patient with major trauma is admitted to a hospital were evaluated by pattern-recognition techniques in an attempt to define an acute prognostic index. The resulting index was a euclidean distance using single pretreatment estimates of arterial pH, hematocrit, prothrombin time, and the systolic blood pressure. Prospective validation of the index resulted in a 98% accuracy of prediction of survival rate with an individual misclassification rate of 15%. When used in conjunction with an anatomical index, the expected misclassification rate was reduced to 8%, and the actual misclassification rate experienced was 3.4%.

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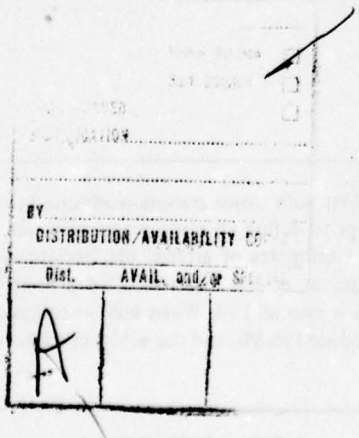
AB

PREFACE

The work described in this report was authorized under Project 3A162110A821, Evaluation of the Care of the Critically Ill Patient; and AMSAA contract DAA D0573C0032. This work was started in April 1974 and was completed in January 1976.



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AN ACUTE-TRAUMA INDEX

I. INTRODUCTION.

An objective quantitation of injury would be of value in epidemiological studies; comparisons of incidence, management, and therapy from center to center; and in defining prognosis. An anatomical scale of injury provides a measure of anatomical disruption. The mortality and morbidity resulting from a given pattern of anatomical injury is significantly influenced by the physiological and biochemical responses that occur as a result of trauma. These responses reflect the severity of injury, the time interval preceding effective treatment, and the effect of age and incidental abnormalities on the individual's protective compensation mechanisms.

Early attempts to quantify this response include those of Cuthbertson in 1930.[1] The predominant effort has been directed toward correlating the degree of trauma and blood loss with measured hemodynamic parameters[2-5] or products of cellular metabolism.[6] More recently mathematical and computer modeling[7,8] have been employed to improve precision and direction of therapeutic management by defining correlations indicative of declining prognosis. The dynamic nature of the physiological and biochemical responses to injury and of the therapies employed explain in part why no reliable index is presently available. The purpose of an acute-trauma index is to characterize, on the basis of a single set of measurements, the acute effects of injury before instituting therapy. This study applied pattern-recognition techniques to parameters commonly measured when a patient with major trauma is admitted, in an attempt to develop a prognostic index reflecting the degree of physiological and biochemical response elicited by acute trauma.

II. METHODOLOGY.

The population studied consisted of patients referred to the Maryland Institute for Emergency Medicine (MIEM) by paramedical triage for suspected life-threatening injury.

Eight physicians from a variety of specialties who were involved in the daily care of the critically injured patient were asked which biochemical and physiological parameters they thought changed most rapidly following injury and which also indicated the severity of injury. Thirty-nine parameters were obtained in response.

III. MEASUREMENT EVALUATION.

The 39 parameters were evaluated, both individually and in combinations of 2 to 12, for their ability to predict the outcome in terms of survival. The first measurement of each variable after admission, before treatment, was recorded on 500 acute admissions. Separation-to-spread ratio, confusion probability, and information gain[9,10] were computed as measurements of the power of prediction for each variable.

Eigenvector mappings and discriminant-plane mapping of select groups of variables were also evaluated with the use of the same three measures. Most analyses were performed by means of the on-line pattern analysis and recognition system (OLPARS) at Rome Air Development Center, Rome, New York.

Results of these analyses were evaluated for ease of potential routine clinical application. Fifteen variables were selected for further structure analysis.

IV. STRUCTURE ANALYSIS.

A retrospective study was carried out on 360 consecutive patients admitted directly from the scene of acute trauma by helicopter or ambulance. Values of the variables on admission, before therapy commenced, were recorded. One to six variables were grouped to form indices on the basis of multivariate analyses (including discriminant plane mappings, eigenvector mappings, and nonlinear distances indices) and on advice from the clinicians.

Each index was evaluated using information gain. This number, which measures the predictive power, can be simply interpreted as the average amount by which one would alter the prognosis of a patient group when provided with a given value for an index or parameter. Mathematically, the information gain g is written

$$g = \sum_{i=1}^{n} |p_D - p_{D,i}| p_i$$

where p_D is the *a priori* probability of death, $p_{D,i}$ is the probability that a patient will die with an index value of i, and p_i is the probability that the index assumes a value i. The range of values of the index is divided into n intervals for the calculation. The maximum value of information gain for a patient population with an *a priori* probability of death p_D is $2p_D(1 - p_D)$. If an index were a perfect discriminator in a treatment facility where the death rate was 50%, the maximum information gain would be 0.50.

On the basis of these computations and the practicality of routinely measuring certain parameters, the acute-trauma index was derived.

V. ACUTE-TRAUMA INDEX.

The acute-trauma index t is based on admission values of systolic blood pressure P, hematocrit H, arterial pH A, and prothrombin time T. The index t is the square root of the sum of the squares of the deviations (measured in standard deviation units) from normal average values for each of the four variables:

$$t = \sqrt{\left(\frac{P - 127}{21.0}\right)^2 + \left(\frac{H - 37.0}{6.0}\right)^2 + \left(\frac{A - 7.46}{0.065}\right)^2 + \left(\frac{T - 13.0}{2.0}\right)^2}$$

Mathematically, this quantity is called the euclidean distance and reflects the difference between an actual patient state and a desired patient state. In each of the squared terms in the sum under the radical, the numerator is the measured value of the variable minus the estimated normal average value, and the denominator is the estimated standard deviation of that variable. The estimates were obtained from the final recorded values from 350 survivors.

The acute-trauma index was used to provide a characterization of a patient at the time of admission and was correlated with patient mortality, up to 6 months following admission, in both tabular and graphic format. The tabulations assign a probability of death to each of several intervals of index values.

VI. SMOOTHING CORRELATION.

The graphical format consists of probability-of-death curves obtained by fitting the data to a logistic model of the form

$$p_D(\mathbf{x}, \boldsymbol{\beta}) = \frac{1}{1 + e^{-A}}$$

where

$p_D(\mathbf{x}, \boldsymbol{\beta})$ = the probability of death

$A = \beta_1 + \beta_2 x_2 + \ldots + \beta_n x_n$

$\mathbf{x} = (1, x_2, \ldots x_n)$ is a vector of measurable variables

$\boldsymbol{\beta} = (\beta_1, \ldots, \beta_n)$ is a vector of weights (coefficients) associated with the measurement variables

Measurement variables may be physiological or biochemical parameters, various indices, age, and may include "indicator" variables (e.g., for sex, 1 = males and 2 = females). The weights were obtained by the Walker-Duncan regression algorithm,[11] which produces approximate maximum-likelihood estimates. In the regression calculation, the dependent variable $p_D(\mathbf{x}, \boldsymbol{\beta})$ used in estimating $\boldsymbol{\beta}$ was assigned a value of 0 if the patients lived and a 1 if death ensued. Probability-of-death curves were obtained for the acute-trauma index alone and in combination with age and sex.

Approximate 95% confidence bounds on the curve were computed by the method of Kendall and Stuart.[12] The model was then tested by applying it to prediction.

VII. PROSPECTIVE VALIDATION OF THE ACUTE-TRAUMA INDEX.

During a 6-month period in 1974, data were collected and computed on 241 acute-trauma victims admitted directly from the scene of the accident. Pretreatment values for the parameters involved in the acute-trauma index were obtained. For each individual patient the acute-trauma index was computed, and the associated probability of death was read from the curves obtained in the restrospective study. The expected number of deaths was computed by summing the probabilities of death and was compared with the actual number of deaths.

A decision rule, which predicted survival if the probability of death was less than 0.5, was used as a basis for the prediction of individual survival. Misclassification rates were then computed for the patient set, and compared with a random-decision rule.

The random-decision rule (RDR) was based on an *a priori* probability of death p_D for the patient population studied. It predicts death for a patient if a random number r, chosen from a uniform distribution of numbers on the unit interval, satisfies $r \leq p_D$. If $r > p_D$, the RDR predicts survival. The expected death rate associated with the RDR would be p_D, and the probability of misclassification p_m would be $2p_D(1 - p_D)$. This latter quantity is obtained as follows:

$$p_m = p_{l,s} + p_{g,d}$$

where $p_{l,s}$ is the probability that $r \leq p_D$ and the patient survives and $p_{g,d}$ is the probability that $r > p_D$ and the patient dies. Because

$$p_{l,s} = p_D(1 - p_D)$$

$$p_{g,d} = (1 - p_D)p_D$$

p_m is, then, given by

$$p_m = p_D(1 - p_D) + (1 - p_D)p_D = 2p_D(1 - p_D)$$

The anatomical index was calculated for each patient.* Predicted death rates and expected and actual misclassification rates were computed using the methodology already described. Records of all patients who were misclassified by either the anatomical or acute trauma indices were individually scrutinized for reasons for the failure.

The data set was further studied to evaluate the use of the two indices in combination. The predicted death rate and expected and actual misclassification rates were computed. For each patient, the probability of death was taken to be the higher probability of death predicted by the two indices if either one had indicated a probability of death greater than 0.5, and it was taken to be the lower of the two probabilities of death predicted by the indices if neither were greater than 0.5. These maximum probabilities were summed to provide the number of deaths.

VIII. RESULTS.

The initial parameters studied and those used for the structural analysis are shown in the following list:

Systolic arterial pressure**
Diastolic arterial pressure**

*Champion, H., Sacco, W., and Ashman, W. Quantitation of Anatomical Injury in Multiple Trauma. Chemical Systems Laboratory Technical Report to be published.

**Parameter chosen for further structural analysis.

Mean arterial pressure
Central venous pressure*
Pulse rate
Rectal temperature
Cardiac output
Peripheral resistance
Pulse pressure*
Hemoglobin
Hematocrit*
Coagulation time
Serum fibrinogen
Serium sodium*
Respiratory rate*
Serum pyruvate
Urine osmolality
Urine urea
Serum water
Prothrombin time*
Serum potassium*
Serum chloride*
Serum osmolality
Arterial pH*
Arterial O_2 pressure*
Arterial oxygen saturation
Arterial CO_2 pressure*
Blood urea nitrogen
Serum Creatinine*
Serum magnesium
Total protein (plasma)
Arterial ammonia
Venous ammonia
Venous O_2 pressure
Serum glutamate oxaloacetic transaminase
Serum glutamic-pyruvic transaminase
Lactic dehydrogenase
Creatine phosphokinase
Serum lactate*

They are 39 in number. Of these, 15 were chosen for further structural analyses. (These parameters are marked with asterisks.)

The probabilities of patient death associated with ranges of values for the acute-trauma index in the retrospective study are shown in table 1, indicating a significant change in probability of death with an index value of greater than 4.0. The coefficients for the logistic model appear in table 2 for both the acute-trauma index alone and in combination with age and sex. Neither of these

*Parameter chosen for further structural analysis.

Table 1. Values of the Acute-Trauma Index Correlated With Probability of Death p_D in 360 Acute-Trauma Victims Studied Retrospectively

Index value	p_D
	%
0 - 1.99	0.06
2.0 - 2.99	.13
3.0 - 3.99	.20
⩾ 4.0	.65

Table 2. Logistic Model Coefficients for the Acute-Trauma Index

Type of index	Constant term	Coefficients		
		Age	Sex	Index
Acute trauma index alone	-3.51	–	–	0.766
Acute trauma index with age and sex	-3.51	.009	-.168	.732

latter parameters had coefficients that were statistically significantly different from 0 and, thus, had no significant effect on the probability-of-death values obtained from the acute-trauma index. The mortality curve for the acute-trauma index is shown in figure 1. The standard error of estimate for this curve rises from 0.01 at an index value of 1, to 0.03 at an index value of 5, and to 0.05 at an index value of 10.

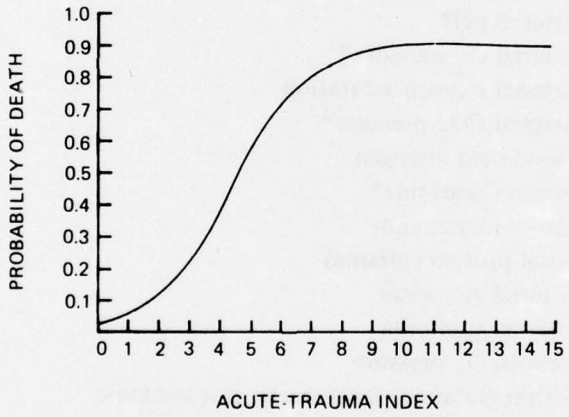

Figure 1. Probability-of-Death Curve for Acute-Trauma Index

IX. PROSPECTIVE VALIDATION OF THE ACUTE-TRAUMA INDEX, OF THE ANATOMICAL INDEX, AND OF BOTH COMBINED.

Of the 241 patients studied prospectively, 45 (19%) died. The acute-trauma index predicted 49 deaths and the anatomical index, 63 deaths. There is an overall accuracy of prediction of expected death rate for this patient population of 98% for the acute-trauma index, 93% for the anatomical index, and 98% for the two indices combined.

When the decision rule was applied to individual patients, both the acute-trauma and anatomical indices had significant information gain, but failed to accurately predict the outcome in a number of patients (table 3), resulting in misclassification rates of 15% and 6%, respectively. Twenty-five of the 37 "failures" in the acute-trauma index were patients with fatal head injuries who were accurately predicted by the anatomical index. Five of the 13 patients who were misclassified (predicted to live) by the anatomical index had remedial surgical injuries, were admitted over 90 minutes after the accident in severe hemorrhagic shock, would have benefitted from earlier therapeutic intervention, and were identified by the acute-trauma index. When the combined indices were used as a predictor of death, the individual misclassification rate was reduced to 3.4%. Seven patients were predicted to live, but died. Of these, one had a brain stem contusion, three died of late sepsis, and three received inadequate resuscitation and surgical therapy. One patient was predicted to die by both indices but lived despite severe multiple-system injury and shock. With the use of the random-decision rule, the probability of misclassification was 30%, which can be compared to the expected and actual misclassification rates in table 4.

Table 3. Misclassification Rates of Indices in Prospective Study of 241 Acute-Trauma Admissions

Index used	Number predicted to die but lived	Number predicted to live but died	Misclassification rate	Information gain
			%	
Acute trauma	5	32	15	15
Anatomical	1	13	6	16
Combined	1	7	3.4	18

Table 4. Expected and Actual Misclassification Rates and Improvement of Actual Misclassification Rates Over a Misclassification Rate of 30% Based on a Random-Decision Rule

Index used	Expected misclassification rate	Misclassification rate of RDR minus expected misclassification rate	Actual misclassification rate
	%		
Acute trauma	14	16	15
Anatomical	10	20	6
Combined	8	22	3.4

X. DISCUSSION.

There have been numerous attempts to quantify the effect of injury on the physiological and biochemical processes. Hemorrhage and hypoxia as a result of trauma cause a series of changes at organ, cellular, and intracellular levels reflecting both the severity of the trauma

and the efficacy of the homeostatic mechanisms of the organism. Any index that purports to measure the acute effects of trauma on the body metabolism is a tentative step toward identifying the slope of the curve shown in figure 2, which can be used to judge both the severity and the rate of change of the reactive process. This concept stresses the dynamic aspects of shock and the extreme importance of time in its evaluation and management.

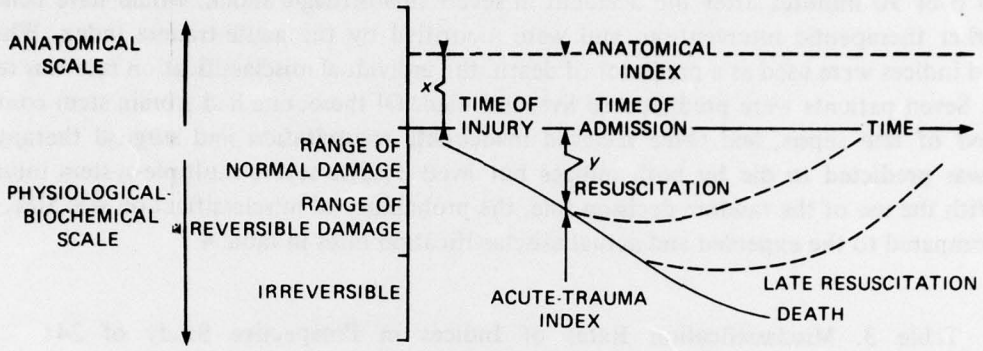

Figure 2. Diagrammatic Representation of the Combined Use of an Anatomical Index and an Acute-Trauma Index, Which Attain Values of x and y, Respectively, Between Injury and Treatment

The approach in this study has been motivated predominantly by statistical rather than clinical methodology. The results may appear bizarre to the practicing physician, who would consider it improbable that a single prothrombin time would enhance his evaluation of a patient critically ill from multiple sites of hemorrhage. The expert appraisal of shock is a difficult clinical challenge frequently associated with delayed diagnosis, misinterpretation of priorities, and a failure to institute appropriate therapy. Unnecessary deaths not infrequently result from clinical deficiences.[13] An approach such as that outlined, which uses sophisticated pattern-recognition techniques, may well contribute to a physician's judgment and accelerate the initiating of therapy. The techniques described are applied to a variety of scientific disciplines, but are still regarded in medicine with an attitude reminiscent of the suspicion that greeted the introduction of the "numerical method" in 1836.[14] Many issues in medicine can be formulated as pattern classification problems with definition of pattern space and decision regions similar to the prognostic regions presented in table 1.[15-20]

The results suggest that the acute-trauma index can accurately predict the outcome of patients with suspected multiple system injury on the basis of a single pretreatment blood sample and measurement of blood pressure. An overall prediction accuracy of 98% with an information gain of 15% would indicate that a further evaluation of patients subjected to alternative processes of triage should be considered. Early death following trauma is essentially a result of acute cellular hypoxia, with late death frequently occurring as an ultimate result of hypoxia or ischemia on vital organ function. The acute-trauma index identifies the majority of patients dying from these causes. Ideally, the slope of the probability curve in figure 1 would be steeper, indicating an abrupt change at the boundaries of the prognosis regions.

The failure of the index to predict death in patients with severe central nervous system injury, who are not in hypovolemic shock, emphasizes one deficiency. To improve the prognostic accuracy, different parameters may require consideration.

The ability of the anatomical index to compensate for these failures and of the acute-trauma index to identify some patients with protracted hemorrhage following relatively less severe injury supports the concept of the combined use of these indices (figure 2). The data indicate a considerable degree of accuracy for individual patient prediction when both indices are considered. The misclassification rates of 6% for the anatomical index alone and of 3.4% for the combined indices are, we suspect, fortuitous, because these values are less than the expected misclassification rates of 10% and 8%, respectively. Random samples in future studies may be expected to approach these latter figures.

The data used were from patients with multiple injuries, and the index attempts to identify patients that are in risk of dying. Although the standard error of estimates is less with the lower values for the index, the value of the index will also be less in large populations with minor injury. Correlation of the lower values of the index with the incidence of organ failure and morbidity after resuscitation would be instructive and may increase the value of the index for patients who survive after resuscitation.

As with other indices reported, the acute-trauma index and the anatomical index would aid in the evaluation of health care and the comparison of institutional management. Subsets of 200 similar patients would be sufficient to compare efficacy of health-care delivery, with confidence limits of 95%, thus providing stimulus for a continued search for reliable, objective, easily applicable indices to characterize both illness and therapy.

LITERATURE CITED

1. Cuthbertson, D. P. The Disturbance of Metabolism Produced by Bony and Non-Bony Injury, With Notes on Certain Abnormal Conditions of Bone. Biochem. J. *24*, 1244 (1930).

2. Bull, J. P. Circulatory Responses to Blood Loss and Injury. Prog. Surg. *4*, 35 (1964).

3. Allgower, M., and Burri, C. Schockindex. Dtsch. Med. Wochenschr. *92*, 1947 (1967).

4. Grant, R. T., and Reeves, E. B. Observations on the General Effects of Injury on Man. Med. Res. Counc. (G. B.), Spec. Rep. Ser. *SRS-277*, London (1951).

5. Burri, C., Menkenmeyer, H., Passler, H. H., and Allgower, M. Evaluation of Acute Blood Loss by Means of Simple Parameters. Prog. Surg. *11*, 109 (1973).

6. Weil, M. H., and Afifi, A. A. Experimental and Clinical Studies on Lactate and Pyruvate as Indicators of the Severity of Acute Circulatory Failure (Shock). Circulation *41*, 989-1001 (1970).

7. Siegel, J. H., Farrell, E. J., Miller, M., Goldwyn, R. M., and Friedman, H. P. Cardiorespiratory Interactions as Determinants of Survival and the Need for Respiratory Support in Human Shock States. J. Trauma *13*, 602-619 (1973).

8. Champion, H., Sturdivan, L., Nolan, J., Stega, M., Cowley, R. A., Sacco, W., and Gill, W. A Mathematical Model for Volume Replacement in Hemorrhage. J. Surg. Res. *19*, 297 (1974).

9. Sammon, J. Interactive Pattern Analysis and Classification. IEEE Trans. Comput. *C-19*(7), 600-610 (1970).

10. Sacco, W., and Copes, W. S. Reduction of the Class of Feature Evaluation Techniques in Pattern Analysis. Pattern Recognition *4*, 331 (1972).

11. Walker, S., and Duncan, D. Estimation of the Probability of an Event as a Function of Several Independent Variables. Biometrika *84*, 1 (1967).

12. Kendall, M., and Stuart, A. The Advanced Theory of Statistics. Vol. I. Hafner, New York, New York. 1969.

13. Gertner, H. R., Baker, S. P., Rutherford, R. B., et al. Evaluation of the Management of Vehicular Fatalities Secondary to Abdominal Injury. J. Trauma *12*, 425 (1972).

14. Double, M. On the Applicability of Statistics of Medicine. Am. J. Med. Sci. *21*, 247 (1837).

15. Weiner, J. M., Hopkins, C. E., Marmoston, J. Use of Correlation Structure Analysis in Estimating Survival Benefit. Ann. N.Y. Acad. Sci. *126*, 743 (1965).

16. Hughes, W. L., Kalbfleisch, J. M., Brandt, E. N., Jr., *et al.* Myocardial Infarction Prognosis by Discriminant Analysis. Arch. Intern. Med. *111*, 338 (1963).

17. Cullen, D. J., Civetta, J. M., Briggs, B. A., and Ferrara, L. C. Therapeutic Intervention Scoring System: A Method for Quantitative Comparison of Patient Care. Crit. Care Med. *2*, 57 (1974).

18. Norris, R. M., Caughey, E. E., Deeming, L. W., *et al.* Coronary Prognostic Index for Predicting Survival After Recovery From Acute Myocardial Infarction. Lancet *2*, 485 (1970).

19. Bleich, H. L. Prognosis by Calculation. N. England J. Med. *285*, 1533 (1971).

20. Afifi, A. A., Sacks, S. T., Liv, V. Y., and Weil, M. H., Accumulative Prognostic Index for Patients With Barbiturate Glutethamide and Meprobamate Intoxication. N. Eng. J. Med. *285*, 1497 (1971).

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